

Conclusion: (1) Image quality and severity of induced dyssynergy have the most significant impact on IOA. (2) Standardization of image display and RC results in improved IOA on DSE interpretation.

1021-121 Accelerated DSE: Is It Safe in High Risk Patients?

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Dobutamine stress echocardiography (DSE) commonly uses 3 minute (min) stages despite requiring up to 10 min to reach steady state. Prior studies have demonstrated the utility of prolonged stage duration, but DSE is already a time consuming test. We developed a novel accelerated DSE (ADSE) protocol in which a single, high dose (40 mcg/kg/min) infusion of dobutamine is administered for up to 10 minutes, or until target HR (85% predicted max) is reached. If target HR was not reached, atropine (0.2 mcg/min, 1.0 mg max) was added. Feasibility, safety, and efficacy of ADSE were assessed in patients (pts) with a high pretest likelihood of CAD. HR, BP, ECG and symptoms were assessed at 1 min intervals throughout the infusion. ADSE was administered to 47 consecutive pts (46 males, 1 female) with a mean age of 64 ± 11 years. Patients had an average of 4.4 ± 1.3 cardiac risk factors and 49% had known CAD.

Results: ADSE increased the HR (68 ± 2.7 to 133 ± 3.9 bpm, $p < 0.01$) with an average test duration of 11.4 ± 3.1 min. Target HR was reached in 19 of 22 (86%) pts not on beta-blockers (BB) but in only 11 of 25 (44%) pts on BB ($p = 0.001$). Symptoms included palpitations (21%), dizziness (12%), chest pain (6%), headaches (6%), or nausea (6%). There were no episodes of sustained VT, VF or serious adverse events.

Conclusions: ADSE with a continuous high dose is feasible and safe in pts with known or suspected CAD and achieves target HR with a shorter duration. Further investigation with an angiographic gold standard will be necessary to determine the sensitivity of ADSE.

1021-122 Optimum Endpoints for Dobutamine Atropine Stress Echocardiography

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Traditional endpoints (ENDPT) for terminating dobutamine atropine stress echocardiography (DASE) have been the use of a predefined maximal dose of dobutamine (DOB) and atropine (AT) or attainment of 85% of maximum age predicted heart rate (MPHR), a value derived from exercise stress testing. Whether these constitute optimum ENDPT for DASE is unclear.

100 consecutive positive DASE studies performed with usual ENDPT (85% MPHR, 40 to 50 mcg/kg/min DOB + maximum 2 mg AT, extensive wall motion abnormality, hypotension, or arrhythmia) were evaluated to assess HR, %MPHR, wall motion score index, DOB and AT doses when positivity of the study (worsening of wall motion by ≥ 1 grade) first developed and at peak dose. Hypothetical ENDPT for peak HR and %MPHR were then applied to these 100 DASE to determine optimum DASE positivity for ischemia. At first positivity, dose of DOB was 28.8 ± 10.9 mcg/kg/min (range 5 to 50) and AT 0.20 ± 0.47 mg (range 0 to 2) vs. DOB 36.5 ± 8.1 mcg/kg/min and AT 0.5 ± 0.7 mg at study completion.

HR (bpm)	%DASE +	%MPHR	%DASE +
110	73%	70	70%
115	83%	75	90%
120	96%	80	95%
130	99%	85	93%

^p $p < 0.001$ vs 110 and 115 bpm. [†] $p < 0.001$ vs 70% MPHR

There was no significant difference in DASE positivity utilizing ENDPT of HR 120 bpm vs. 75% MPHR. At traditional ENDPT of 85% MPHR or maximum DOB and AT dose, diagnosis was changed from single vessel CAD to multivessel disease in only 6 studies.

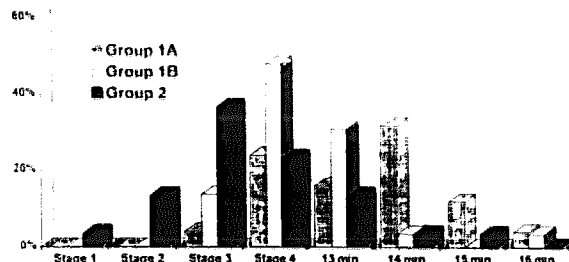
In conclusion, optimum ENDPT for DASE include HR 120 bpm or 75% MPHR in addition to ischemia, hypotension, or arrhythmia.

1021-123 Use of Atropine Early During Dobutamine Stress Echocardiography

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A significant percent of pts undergoing dobutamine stress echocardiography (DSE) will fail to reach target heart rate with dobutamine alone and will require atropine (ATR). We have previously defined criteria which predict failure to reach $\geq 85\%$ PMAX by 12 min (end of Stage 4 - 40 mcg/kg/min) of standard

DSE protocol. These criteria are Age > 50 yrs OR HR at baseline < 60 bpm OR HR at end of 6 min (Stage 2 - 20 mcg/kg/min) $< 60\%$ PMAX. 54 pts meeting these criteria were randomly assigned to either a standard McNeill protocol (in which 0.25 mg ATR is given q 1 min after completion of Stage 4 to a total dose of 1 mg) (Group 1A, $n = 25$) or early atropine protocol (Group 1B, $n = 29$) in which 0.25 mg ATR was given at the beginning of Stage 3 and of Stage 4, with an additional 0.25 mg ATR given at 13 and 14 min if needed. These patients were compared additionally with 30 pts who were predicted to achieve $\geq 85\%$ PMAX and received a standard McNeill protocol (Group 2). Time to peak HR was shorter in Group 1B than in Group 1A (12.0 ± 0.3 vs 13.7 ± 0.3 min, $p = 0.001$), although still longer than in Group 2 (10.4 ± 0.5 min, $p = 0.01$). In addition, Group 1B and Group 2 had similar rates of decline in HR after study termination, reaching $66 \pm 1\%$ and $68 \pm 2\%$ PMAX respectively at 6 min post. HR in Group 1A remained higher, longer, falling to only $72 \pm 2\%$ at 6 min post ($p = 0.01$). Graph below shows stage at which pts reached peak HR.



Conclusions: 1) Early atropine use significantly shortens DSE protocol. 2) HR returns to baseline values quicker with early atropine administration.

1021-124 Pulmonary Artery Systolic Pressure Response to Exercise: Defining Physiologic and Pathologic Limits

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Kinetic exercise increases the cardiac output and pulmonary blood flow and consequently pulmonary artery systolic pressure (PASP). However the upper physiologic limit of PASP response to exercise has not been completely addressed. The objective of this study was to define the spectrum of PASP during exercise.

Methods: From 1994 to 1997, 102 subjects (mean age 36.9 ± 15.6 , range 21 to 80 years) (56 men, 46 women) underwent a standardized semirecumbent echo Doppler bicycle exercise. PASP was calculated by the formula: $4 \times \text{tricuspid regurgitation velocity}^2 + 14$ mmHg (right atrial pressure). There were 40 normal controls (I), and 62 patients (II) with known or suspected pulmonary hypertension referred to our institution for testing.

Results: The 95% confidence intervals (CI) for mean PASP were calculated for normal controls (table below). The upper limit (mmHg) for normals was:

Watts	Rest	40	80	120	160	200	240
Upper CI limit	35.8	42.1	45.8	48.9	60.8	59.3	67.3

Of the patients in group II 49 had PASP at rest and at each stage $> 95\%$ CI seen in normals, 8 had exercise induced PASP (ExPASP) and 5 had normal PASP at rest and during exercise.

Conclusion: (a) Physiologic range of PASP during stress is higher than previously recognized and provides a standard for the diagnosis of pulmonary hypertension. (b) The presence of ExPASP confirms that resting normal PASP does not exclude occult abnormalities of the pulmonary vessels.

1022 Echocardiographic Measures of Left Ventricular Function and Flow

Sunday, March 29, 1998, 5:00 p.m.-7:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 5:00 p.m.-7:00 p.m.

1022-137 Effect of Rhythm on Interpretation of Digital Echocardiograms

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Background: We have shown that digital echo (DE) is a reliable substitute